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INTESTINAL OBSTRUCTION

A Survey of the Etiology, Pathology
and Treatment

by

Sherman S. Pinto

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ILEUS

Ileus, or intestinal obstruction is interruption of the fecal current in the intestines. The types of ileus depend on the etiological factors. They are: (1) reflex ileus; (2) mechanical ileus; (3) inflammatory ileus; (4) dynamic, spasmodic, or spastic; and (5) adynamic paralytic, or inhibitory. (4).

ETIOLOGY

A. Neurogenic. Ileus may be caused by either direct nerve injury, or the intestines may be affected through a reflex mechanism. In the direct nerve injury type, it is injury to the afferent system which is of prime importance. The cord may be injured either by trauma or disease. A fracture of the spine is the commonest traumatic lesion which produces the condition. Diseases of the cord such as tabes, or myelitis may also affect the afferent system to the intestines, and so produce an ileus.

Certain pathological conditions - both intra-thoracic, and intra-abdominal - may produce an ileus reflexly. It is conceivable that such a mechanism might act by either (1) inhibition of the vagus group; or (2) stimulation of the sympathetic system. The vagus nerve, and Auerbach's plexus normally stimulate peristalsis, while the sympathetic vasomotor nerves inhibit peristalsis. Conditions which are known to produce an ileus through a reflex mechanism are: (1) renal calculi, (2) trauma

to the ovary, (3) a blow to the testicle, (4) a biliary stone, (5) a tumor on a twisted pedicle, (6) pleurisy or pneumonia. It might be said that, except for pneumonia, the occurrence of a paralytic ileus with one of the above conditions is relatively rare.

B. Vascular. Either embolism or thrombosis of the mesenteric vessels will quite frequently produce an ileus.

C. Toxic. Toxins may come from within or without the body to produce an ileus. Such exogenous drugs as lead, tyrotoxin, etc., may effectively stop peristaltic action of the gut. Endogenous toxins which produce an ileus are of a bacterial nature. In a local or general peritonitis there is formed a lymph-adhesive exudate which by both mechanically binding the coils of the intestines together, and by direct toxic action may cause the paralysis of a segment of gut. (48).

D. Mechanical Obstruction. Bands or adhesions may be formed without the gut and produce a mechanical obstruction. Trauma by an unskilled operator is very apt to cause such adhesions. The commonest mistakes, made at operation, which cause bands are rough handling, prolonged exposure of intestines to air, and the use of extremely hot sponges. Tumors without the intestine, or strangulation of a hernia, are also factors external to the intestine which may cause an obstruction.

Bodies within the intestine, such as impacted feces, large gallstones, masses of parasites, or coproliths,

may cause obstruction by a direct blocking mechanism.

In the wall of the bowel, especially the lower bowel, obstructions are usually of an organic type. Annular carcinomas, strictures of healed ulcers or syphilitic lesions, or intussusceptions may prevent the passage of fecal material.

Occasionally, the increase of intra-abdominal tension which may result from the reduction of a large hernia, or from extensive resection of the anterior abdominal wall with closure, may produce an obstruction.

AGE INCIDENCE

In infancy, intussusception is the commonest cause of obstruction. 50 per cent of all intussusceptions occur in children less than one year old. Congenital strictures and bands from Meckel's diverticulum may also be found as etiological factors.

In middle age, the commonest causes of obstruction are strangulation of herniae, adhesions, and annular carcinomae.

In old age, volvulus and cancer are the two most important factors in the production of intestinal obstruction. (4).

PATHOGENESIS AND PATHOLOGY.

A. Essential organs.

In the intestine are naturally to be sought the first evidences of obstruction. The blood vessels of the intestine are markedly congested. Above the point of obstruction, the gut is dilated, and of a dusky red color. The fluid content within the intestine is reddish-brown, foul smelling, and teems with bacteria. In high obstructions, the mucosa from the point of obstruction to the pylorus will be found to be injected, and often ulcerated or necrotic in areas. Microscopically, there is a round cell infiltration to be seen, and petechiae are common. Below the obstruction, the intestine is normal, although collapsed. The peritoneum surrounding the involved segment of intestine is not involved early. (54).

There seems to be a close relationship between the distance of the obstruction from the pylorus and the severity of the symptoms produced by it. Dogs, Roger (75) has shown, lived 24 to 36 hours if the obstruction was in the duodenum. With the obstruction at the mid intestine, they lived 5 to 6 days, and, if the terminal ileum was blocked, death came only after 10 to 12 days. With rectal obstruction some of them lived as long as 28 days.

Some investigators of intestinal obstruction (84) (17) (33) (73) have shown the great similarity of a closed loop to a simple obstruction. In a closed loop, a small segment of intestine is isolated and the ends inverted. The two free ends of the intestine are then united so as to re-establish the continuity of the intestinal canal. However, closed loops in the colon or low in the ileum do not seem to produce any symptoms. In the case of high loops, the length of the loop is of major importance. In general, the shorter the loop, the quicker does death ensue. Not all animals with closed loops show the pathological picture which will be presented here, but they do show enough of it to be significant.

The blood supply of the intestine at the point of, and just in front of, the point of obstruction is a determining factor in the severity of the condition. Strangulation produces death much more rapidly. This has been proven by many workers. (13) (42) (57) (102). A very short strangulated segment becomes, then, the most fatal type of lesion. (13). The toxemia is so acute that there is no time for detoxication of the organism. Of the blood changes (see below) only the urea content rises markedly.

In studying the pathological changes of the intestine, the changes in the properties of the intestinal

contents first attract our attention. Babsky (5), working on cats and dogs, showed the effects of normal intestinal contents on these animals when it was injected intravenously. He introduced distilled water, 0.5% HCl, or physiological NaCl solution into the lumen of the small intestine, withdrew it in 10 minutes, and injected it intravenously. He got (1) stimulation of the motor activity of the small intestines with marked contractions, or (2) an increase in intestinal tonus which gradually returned to normal, or (3) a single strong contraction, or (4) a decrease or inhibition of the contractions with a fall in tonus, or (5) irregular combinations of the above effects. The various effects observed seemed to be dependent on changes in the state of irritability of the peripheral organs. The fluid obtained from the introduction of HCl into the duodenum was the most active. Boiling the fluid at pH of 7.0 did not destroy its activity, but if the pH was less than 7, the activity was destroyed. The effects bore no relationship to blood pressure, and were not affected by cutting the vagi, and the splanchnic nerves. This shows the substance present acted on the muscle or intrinsic innervation of the intestine.

When material from a strangulated gut is injected into a normal animal, the animal dies with a typical symptom complex. There is vomiting, dilatation of the pupils, tenesmus, tremor, and a weak rapid pulse. The urine shows a great increase in N.P.N., most of which

is attributable to urea. Of interest is the appearance of the intestine in the injected animal. The mucosa is red, and velvety in appearance and this change takes place almost universally at a point beginning 1 cm. below the pylorus. Farther down in the intestine is seen a secretion of blood stained fluid. (1) (39) (83) (84). If the intestinal contents of an animal which has died from a toxic injection be used for a similar experiment, they will also be found to be toxic. However, these toxic intestinal contents must be injected intravenously in each case to be effective. If they are given by mouth, they are harmless. (39).

The relation of bacteria to death in high obstruction has been shown by Buchbinder (15), and Helmberger and Martina (56). Early workers felt that organisms migrated through the wall of the intestine and caused a fatal peritonitis. These workers have disproved this contention. Hartwell and his coworkers (54) have shown repeatedly that sterile cultures may be obtained from the intestinal contents of an obstruction, and that bacteria are not the fundamental etiological factor.

The toxic intestinal contents have been prepared in a number of ways for intravenous injection. Murphy et al (73) (25) either used the contents undiluted, or diluted with an equal volume of water. Clairmont and Ranzi (21) filtered through a Chamberland filter and found that the

toxic principle was not removed. Dragstedt, Dragstedt, and Chase (30) strained the fluid, heated it for 1 hour at 70° C, filtered, and preserved the filtrate with chloroform. The chloroform was removed by heating before using, but the toxicity of the filtrate was not destroyed. Stone, Bernheim and Whipple (84) used essentially the same procedure for preparing the intestinal contents except they heated at 60° C for several hours. Ellis (39) precipitated the toxic substance with five volumes of 95% alcohol, extracted the precipitate with boiling water, and salted out the protein with MgSO₄. To the filtrate he added five volumes of 95% alcohol, removed the precipitate, and dialyzed it against distilled water. Even after this treatment, the toxic substance was still very active.

These different methods of treating the intestinal contents which contain the toxic agent make it apparent that there is one or more active substances present which are deleterious to the animal organism. Other workers have observed these facts about the toxic agent. It is apparently absorbed by an earthen filter but is not retained by a paper filter (96). Repeated filtration will destroy the toxicity of the substance (21), as will repeated precipitation (101). A collodion membrane is impermeable to the substance (18), and if put in a collodion bag and dialysed against isotonic NaCl for several hours, the substance will lose its toxicity (24).

Long standing in the icebox will destroy its potency (21), as will hydrolysis at 100° C for six hours with 5 per cent H_2SO_4 (84). However, it is not destroyed by boiling at pH 7 (18), nor by pancreatic digestion in seven days (84). Months of autolysis with intestinal mucosa will not destroy it (97), nor will erepsin (39). From its occurrence, it is seen to be soluble in water (39). It may be thrown out of solution by a solution rich in albumin, but whether this is a direct precipitation or an adsorption phenomenon is hard to say (97). It is precipitated in an 83 per cent alcohol solution (39), and by 60 per cent ammonium sulphate (24).

In considering what substances in the intestinal tract might give rise to such a compound, there are two classes to be considered. First, there are the products of normal digestion, and, secondly, the products of more marked action of bacteria on the products of a stagnant digestive system. In brief, from the numerous compounds which have been suggested, these would seem to be the most likely: split proteins, mercaptans, fragments of amino acids - cresol, phenol, phenylacetic acid, etc., oxidation and reduction products of tryptophane, neurine, muscarine, complex fatty acids, glucosamines, various toxic amines - ptomaines, putrescin, cadaverine ethylenediamine and histamine. (46) (88). Kukula has estimated that none of these substances exist in sufficient quantities in the intestine to cause the observed effects (63).

Koessler (62) has even shown in what enormous quantities a definitely toxic substance, such as histamine is known to be, may be present in the stomach or even the intestinal wall without apparent injury to the animal.

Other workers have attacked the problem by analogy. Certain definite substances produce an action on the organism similar to that produced in intestinal obstruction. Whipple (85), and Mendel and Henderson (20) have shown the striking similarity between the substance and a protease. Gerard (46), and Stone (83) have attempted to identify it with histamine. Barger and Dale (7) showed its similarity in action to peptone, and certain tissue extracts.

Whipple (85) (100) has attempted to show the toxic substance is a protease because of the similarity between the two substances chemically. He even goes so far as to say he has produced relative immunity to the substance. In connection with this point the work of Walzer (94) on the absorption of undigested proteins in human beings is stimulating. He produced much evidence to show that a detectable amount of protein frequently entered the blood stream in an unaltered state by way of the digestive tract. On the other hand, there are numerous investigators who have never been able to produce any signs of immunity. Brooks (13), Ellis (39), Dragstedt (18) (30), Davis and Morgan (27), Ingvaldsen, Whipple, Bauman and Smith (58)

succeeded in getting one gram of white powder which contained, or was, the toxic substance. They thought that from chemical analysis the substance was a nucleoprotein.

Of all the possible sources for the toxic substance - stomach, biliary tract, pancreas or intestinal mucosa - only the intestinal mucosa has not been eliminated by convincing experiments. Within the intestine are found normally the mucoid secretion from the glands of Brunner, the trypsin which has been formed by the action of enterokinase on trypsinogen, carbohydrate-splitting enzymes, muclease, and secretin. The mucosa of the intestine contains also erepsin which under abnormal conditions, such as are found in obstruction, may be forced into the lumen of the gut. Stone, Bernheim and Whipple have attempted to demonstrate that it is the intestinal mucosa which elaborates the toxin (85). Mucosal extracts of animals, which had an obstruction, they found to be toxic. In their opinion, the toxic substance was formed by the mucosal cells, some going into the lumen of the gut, some into the circulation, and some staying in the cells. The theory of action which they built up was this: The normal passage of the intestinal contents was the stimulus for the normal action of the mucosal cells. Interference with this stimulating action caused the cells to produce a toxic or perverted substance. Sweet (87) has criticised their technique in that they pro-

duced a mechanical obstruction and did not prevent the possible formation of a functional obstruction. So, the toxin could have been formed in a section of intestine which was functionally obstructed, and reexcreted into that part of the intestine which they had operated on.

Draper (35) (36) (67) and his co-workers are of the opinion that the toxic substance is a normal product of the duodenum which is neutralized in the lower intestine. Any interference with this neutralizing process would cause the absorption of the unneutralized, or undetoxified, toxin.

Dragstedt et al (34) have done some very significant experiments in which they try to show the dissociation of the intestinal secretion and the toxic substance. Segments of intestine were isolated and left open in the peritoneal cavity. All blood vessels and nerves were left intact. Later, another laparotomy was done and the contents of the isolated segments were found to be sterile. The ends of the segments were then inverted and closed. Animals so treated did not develop symptoms of obstruction, but remained well indefinitely. Strangulation of these segments also did not produce any toxic symptoms. It did not matter at what level the isolated segments were taken from. This would seem to be prima facie evidence that the intestinal

secretion was not the causative agent of the toxemia. By the use of chloroform, Davis and Stone (28) claimed that they eliminated bacterial action from the intestinal contents without affecting enzyme action. Secretions from intestines so treated they found to be non-toxic. Murphy and Brooks (73) also tried to eliminate the effects of the intestinal mucosa in this manner. They tied off the cystic duct in a dog and found it compatible with life. Then, into the distended gall bladder they injected some of the bacteria-containing contents from an obstruction. In a short time, the animals died from a toxemia typical to that observed in the ordinary obstruction. This would seem to show it is not the secretion of the intestinal mucosa which is the toxic agent, but instead it is the product of bacterial activity.

The tremendously varied bacterial flora of the lower part of the intestinal tract is well known. But the duodenum and stomach are often considered to be sterile due to the relatively strong HCl concentration within the stomach. However, MacNeal and Chace (65) have shown that anything which disturbs the chemical or motor functions of the stomach may lead to the growth of micro-organisms in the upper intestinal tract. Davis and Stone (28) showed that as long as bacterial activity was prevented normal intestinal contents would not become toxic. If it was incubated without antiseptic precautions

being taken, it developed toxic properties. Whipple, to the contrary, found putrefied and autolyzed mucosal extract to be non-toxic.

Allbeck (1) found the intestinal canal of hibernating animals to be sterile. He strangulated a long section of gut in such an animal and found no toxemia in two days. At this time the animal awoke and was fed some milk. In six days death from toxemia occurred. The toxemia, he thought, was the result of bacterial action by bacteria which had been introduced by the ingestion of the milk.

Dragstedt (33) (31) (34) hoped to eliminate the effect of bacterial action by sterilizing isolated segments of gut by the use of ether. After such an experiment, many of their experimental animals remained alive for some time. Cultures from these segments at postmortem, however, showed the presence of bacteria in small numbers. According to these workers, the ether exerted an astringent effect on the intestinal mucosa with a resultant reduction in permeability. Other experiments by Dragstedt have been alluded to above in which the isolated segment of intestine was sterilized by other means. It was the result of these, and similar experiments, which caused many students of the problem to come to the conclusion that bacteria were responsible for the toxic agent.

Williams (103) investigated the effect of bacteria in intestinal obstruction with especial reference to the anaerobe bacillus welchii. The report of the Committee on Anaerobic Bacteria which functioned during the World War showed him the striking similarity between the toxemia of intestinal obstruction and that of gas gangrene. They stated: "Vomiting is frequent. The pulse is rapid. The extremities are cold and blue. The temperature falls. The mind stays acute to the end. In the terminal stages, some degree of icterus may be present". By investigation he found B. welchii to be the commonest anaerobe in the intestinal tract. In a large percentage of cases suffering from acute obstruction he was able to isolate the organism. In order to demonstrate experimentally that the toxic substance was B. welchii toxin, he performed an extensive experiment using mice as the experimental animal. Into one series, he injected the contents of the intestine after an obstruction had been produced. Into another series, the toxic intestinal contents were injected along with B. welchii antitoxin. 50 per cent of the first series died, whereas all of the second series lived. On looking further for pathological evidences to support his contention that the toxin of B. welchii was the toxic agent in obstruction, he found other characteristic lesions of B. welchii intoxication. These were fatty degeneration of the liver and heart, and a hemolytic staining of the intimal

layer of the arteries. The next logical step, then, was to apply the therapeutic test. At St. Thomas' Hospital in London he elected to use his treatment in selected cases of acute appendicitis. From a series of 256 cases he chose 18 in which all consultants agreed chances of living were 50 per cent or less. His death rate in these selected cases was 1.17 per cent. In a control series run at the same time, the death rate was 6.3 per cent. Over a period of years, the death rate in acute appendicitis at that hospital has been established as 6.5 per cent. Then, to nineteen cases of acute obstruction he gave the antitoxin. Five so treated died. The mortality rate in the untreated cases from 1919 to 1923 (214) was about two and a half times that of his antitoxin treated cases. Many patients made a striking improvement as soon as they received the antitoxin. On the face of these experiments, they seem rather convincing. Certain objections, however, must be answered. *Bacillus welchii* toxin is very heat labile, and is quickly destroyed by heating. As has been shown before, one of the striking characteristics of the toxin in acute obstruction is its resistance to heat. In a broader sense, all those who uphold the bacterial origin of the toxin do not account for its extremely rapid production both in animals with the obstruction and in animals which have received an intravenous injection of the substance. Another fact which must be explained is the difference in toxicity between a low and a high

obstruction when it is the lower intestine which contains the greatest number of bacteria.

Buchholz and Lange (16) fed animals on acriflavine in such amounts that no bacteria were able to develop in the intestine. This did not influence the effect of obstructing the intestine.

Most investigators have starved their animals before studying the obstruction picture. Others have studied the effects of diet. In general, a meat diet was found to be approximately nine times as toxic as a milk diet. A diet of proteins preceding the obstructive operation assured the operator of quick, toxic results (102).

The effect of the toxin seems to be a systemic reaction. The speed with which it reacts would suggest that it was blood borne. Sauerbruch and Heyde (78) showed this by a parabiosis experiment. If one of two such animals was obstructed, the other showed the toxic effects at the same time. By terminating the parabiosis, the animal which was not obstructed could be made to recover. Murphy and Brooks (73) thought that the toxin left the intestine through the lymphatics, and entered the venous system via the thoracic duct. They put a cannula into the thoracic duct of a dog and collected material. Then, they isolated a segment of intestine, injected material into

it from the intestine of a dog which had died from acute obstruction, and by means of a column of water they increased the pressure within the isolated segment of intestine. Quickly, bloody lymph appeared in the cannula, was collected, and on injection into other animals proved toxic. Normal lymph was non-toxic. Of course, the appearance of bloody lymph in the cannula could only mean that certain normal capillary barriers had been broken down and their experiment does not prove conclusively the route of toxic absorption. Van Zwahlenburg (91) has studied circulatory changes in distended hollow viscera with these results. At 30 mm. Hg pressure some of the capillary streams were arrested. At 60 mm. pressure some of the small veins were arrested and the current was slowed in most of them. At 90 mm. pressure, all blood streams moved slowly, many not at all, and some in reverse. All circulation ceased at 130 mm. pressure.

Morton (72) injected the vessels of the lower duodenum and terminal ileum with a barium mixture, and india ink. A much richer supply of arteries was found in the duodenum than in the ileum. There was also a richer capillary network in the duodenal villi, and a wider, more extensive anastomosis through the duodenal wall. Under the conditions of a closed loop obstruction, the duodenal villi were flattened out so that the capillary loops formed a dense, dilated mat with overdisten-

tion of the vessels immediately below the mucosa. The ileum under similar circumstances showed less effective "ironing" down of the villi and less submucosal engorgement. The secretion rates of the duodenum were compared and it was found that the duodenum secreted from 5 to 10 times more fluid than the ileum in the same period of time. The bursting pressures of equal segments of duodenum and ileum were compared. In general, the amount of force necessary to cause rupture of the segment was approximately the same. Both the duodenum and ileum were found to be able to stretch considerably in length and circumference. The theoretical sequence of events in the development of toxicity in intestinal obstruction as postulated by Morton is this. There is first a latent period of varying duration during which no symptoms are manifest. In this time, fluid is being secreted into the loop and bacterial growth is taking place. Histamine or histamine-like bodies are formed and taken up into the intestinal wall. The capillaries become distended, and the circulation is slowed. The bowel at this stage shows a dusky flush of capillary congestion.

There is a loss of fluid from capillaries into tissue spaces, giving the soggy, edematous bowel found at this time. Alteration in capillary tone leads to stasis and a lack of oxygen. Tremendous osmotic pressure is thus developed toward the capillary loops. The

intra-intestinal gaseous fluid pressure also increases greatly.

Whatever toxins are present in the lumen or mucosal cells may thus be forced into the intestinal walls by some route - blood vessels, lymphatics, directly through cells, or intercellular spaces - the exact mechanism being still open to question. Fisher and Moore (41) have shown that by changes in the blood salts, the cells of the intestinal mucosa can be altered so as to become permeable to substances to which they were once impermeable. The toxins remain in the wall which has a circulatory and lymphatic stasis so that they cannot be moved. They are retained as in a sponge. With increasing intra-intestinal pressure, the toxins are eventually squeezed out into the systemic circulation.

Necrosis is probably not necessary, but may accelerate the process by exposing the capillary loops to direct action of the poisons. The injections of india ink showed that the capillary loops remain intact even under considerable intra-intestinal pressure. It would seem strange if the defensive reaction of the body were to fall down, and not wall off absorption from a necrotic area by thrombosis. When necrosis goes all the way through the wall, however, absorption becomes possible from the peritoneal side, and rupture of the loop with rapidly fatal peritonitis gives another angle to the picture.

Werelius (96), and Clairmont and Ranzi (21) tried transfusing the entire blood content of a toxic animal into a normal animal. There were no toxic symptoms produced. Scholefield found it was only in the few minutes preceding death that the blood of the mesenteric veins became capable of producing toxic symptoms.

It was stated above that the normal intestinal mucosa was impermeable to the toxic principle in intestinal obstruction. There can be no doubt that with an obstruction there is a distention of the gut, and an increase in intra-luminal pressure. Thus, it is not hard to imagine an injury to the mucosal layer also takes place. The microscopic picture of such an intestinal mucosa is given above. Stone and Firor (86) found the normal pressure within the intestine to be 1.5 cm. water. In a case of obstruction, the pressure within a distended gut often reached 150 cm. water.

A number of workers have started with the assumption a priori that the mucosa is injured by an obstruction, but the rate of absorption through it differs from the normal. In order to test this, they have used such substances as strychnine (12), calcium iodide (21), and phenolsulphonphthalein (26). Whether absorption was increased or decreased seemed to depend on the substance tested, since their results were at such variance.

Brooks et al (79) (44) (102) have worked out the most likely theory concerning injury to the mucosa. They thought that the degree of toxemia was directly proportional to the degree of injury to the mucosa. The injury, they believed, was the result of distention of the gut, and to interference with the circulation. The upper intestine is largely secretory, so it becomes distended much more rapidly than the lower intestine when obstructed. They state that the toxin is probably bacterial in origin, and its absorption is greatly increased by injury to the mucosa.

Van Buran (90) found that the mucosa showed microscopic evidence of injury about 48 hours after production of the obstruction. It is at this time that evidences of toxemia first appear. Clinical experience has taught that after 48 hours of obstruction, surgical intervention is of little use. This substantiates the data of experimental workers.

Again, it should be reiterated that the exact route the toxin follows after it passes through the mucosa is unknown. Since strangulation of the mesenteric vessels is so rapidly fatal, it does not seem likely that the toxin goes directly into the portal system, but rather goes first to the thoracic duct. (73).

Some investigators have studied the possibility of the toxic substance going through the intestinal

wall and being taken up by the peritoneum. Braeye (11) looked upon the intestinal wall as a semipermeable membrane. If it was, he thought he should be able to prove it by experiments of osmosis. He knew a collodion membrane - ordinarily impermeable to hemoglobin - could be made permeable to the passage of hemoglobin by the addition of sodium oleinate, and sodium glycocholate to the solution. So, into a Ringer's solution plus 1 per cent soap, he put an excised, closed loop which was full of toxin. After a suitable interval, he was unable to demonstrate any toxicity of the Ringer's solution.

Schonbauer's experiments seem more physiological since he did not remove the intestine, which was to be tested, from the peritoneal cavity (80). His experiment was this: Calcium iodide was put into the stomach per os. In a period of twelve hours he could find no evidence of it in blood, urine, or peritoneal cavity. Then he repeated the experiment except that, after placing the calcium iodide in the stomach, he did an exploratory laparotomy and put a solution of silver acetate in the peritoneal cavity. The abdomen was closed. In six hours the dogs became very sick. At this time, the blood and urine were iodide free, but on opening the peritoneal cavity he found a large amount of silver iodide and even an excess of free calcium iodide. Using the same experimental method, a passage of calcium iodide into the peritoneal cavity was found in the case

of both simple obstruction and strangulation. Testing the passage of other substances through the intestinal wall, it was found that crystalloids went through much more rapidly than colloids. Bacteria were unable to pass through the intestinal wall at any time. These experiments show rather convincingly that even the simplest abdominal operations affect the permeability of the intestine.

Only the question of the possible formation of the toxin by the mucosal cells has not been subject to extensive experimentation. This, of course, is due to experimental difficulties. Davis (26) by a process of elimination attempted to show that the normal contents of the upper intestine were toxic, and that through some obscure mechanism the action of the mucosal cells was so perverted as to secrete this substance into the blood stream.

To summarize, we may say that the toxin is formed in the intestine or mucosa, and by some means reached the blood stream where it is carried to the various organs.

Physiological research has shown that the vagal impulses are augmentors, and sympathetic impulses are inhibitory to intestinal peristalsis. (60) (68). Thus, it is evident that three possible neural changes may give rise to obstruction in some portion of the

intestinal tract. These are: (1) paralysis of both systems; (2) relative paralysis of the vagus, or overaction of the sympathetic, for all impulses; (3) relative paralysis of the vagus for those impulses which pass only to the sphincters.

Paralysis of both systems seems to be a personal idiosyncrasy, so that certain individuals under slighter strain than is usually required will develop paralytic dilatation of the stomach or intestines. At the operating table, the three most potent predisposing factors seem to be: administration of an anaesthetic, rough handling of the intestines, or the presence of sepsis. The stomach becomes immensely dilated, and the wall is so thin that the contents within may be seen. This thinning and dilatation extends well into the duodenum, gradually fading away in the upper jejunum. The normal mucosal folds are flattened, and areas of superficial ulceration and erosion are constant. It is the loss of blood in small quantities from these areas which gives the brown or black color to the vomitus. (93).

No local peristaltic waves are to be observed in the affected area, and this is further shown by failure to obtain relief from drainage. The exact site of the lesion would appear to be in the nerve pathway since adrenalin given by mouth acts directly on the gastric muscle causing it to contract. Koehler (61) believes the condition is one not of complete paralysis, but rather is a balance

of the two nerve systems at a lower level than normal. He showed that injection of the coeliac plexus with nicotine would paralyse the sympathetics and peristaltic movements would start immediately. Spinal anaesthesia has been used for a similar reason with good results in selected cases.

Relative paralysis of all vagal impulses is a condition which has not been proven, although there is considerable suggestive evidence of such a change which would fully explain the clinical symptoms. The condition is characterized by delay in emptying, by dilatation, and by the absence of any visible obstruction or of hypertrophy of the wall behind the suggested obstruction. In many cases there are definite areas of spasm. X ray examination may show a notch of depression on the greater curvature of the stomach, or the whole pyloric region may be involved. This condition not infrequently occurs as a reflex phenomenon with lesions situated in other portions of the abdomen, such as the appendix or gall-bladder, or more commonly with gastric or duodenal ulcers.

A mild degree of dilatation and apparent atony of the duodenum is common in all viscerotonic cases. Since the symptoms date back to childhood, there is suggestive evidence that the condition is congenital in origin. Most of these cases at operation show a slight degree of ulceration at the position of the superior

mesenteric vessels, but there is no muscular hypertrophy which would suggest a mechanical obstruction.

Colonic spasm occurs most often in those about 40 years old. The affected portion of the intestine shows no evidence of mechanical obstruction and no hypertrophy of the gut behind the areas of spasm. Mucous colitis has been found associated with about 25 per cent of such cases by Ryle (77).

In those cases in which there is a relative paralysis of the vagal impulses to the sphincters an enormous dilatation and hypertrophy of a portion of the intestinal canal is seen. Post-mortem examination shows no obstruction, although the change is sharply limited below, usually at the level of one of the fixed sphincters. No microscopic change can be demonstrated in the muscle of the sphincter.

In Hirschsprung's disease there is an enormous dilatation of the colon with hypertrophy of the muscular wall. No mechanical obstruction is present. As a general rule, the whole colon from the rectum to the caecum is involved. Occasionally, the muscular wall may be one-half inch in thickness. During life, active peristaltic movements are visible. Above, the change may extend to and be sharply limited by the ileo-caecal valve, or may cease lower down. Below, the dilatation usually ceases at the pelvi-rectal junction,

either sharply or by a funnel shaped narrowing. The anal canal rarely shows dilatation. Since no obstruction is to be found post-mortem, and there is such an hypertrophy of the gut wall, there is a growing tendency to accept the view that the lesion is a neuromuscular fault. The ease with which enemata may be administered is further support of the view that the condition is due to a relative paralysis of the vagal impulses to the sphincter (93).

B. Associated Lesions.

1. Blood and Bloodvessels. The only demonstrable change which occurs in the blood vessels is an hemolytic staining of the intima. Williams (103) is the only observer to report this.

Changes in the chemical composition of the blood itself are not as marked in cases where there is strangulation of a segment of the intestine. In these cases, the overwhelming toxemia seems to kill the organism before changes in the blood have taken place. There is, however, a rise in blood urea (22). No one has reported any changes in the blood chlorides or the CO₂ combining power.

Tileston and Comfort (89) were the first investigators to describe a rise in blood urea in cases of intestinal obstruction. Cooke, Rodenbaugh and Whipple (22) studied this rise and stated that in their series

the rise was proportional to the severity of the symptoms. The rise in the blood urea may be a result of decreased kidney function, of increased urea formation, or a combination of both. Haden and Orr (49) answered this question when they showed the excretion of nitrogen into the urine was about five times the normal amount. This shows the rise in blood urea is due mainly to increased formation. McQuarrie, Irvine and Whipple (69) determined kidney function by excretion of phenolsulphonphthalein, and found it was slightly diminished. Other evidences which they found of partially decreased kidney function was an albuminuria, and a small increase in blood creatinine. Thus, there is no evidence of a greatly damaged kidney.

Urea is formed mainly from products of protein metabolism. It would seem, then, that there is some agency causing an increase in protein metabolism. Whipple has expressed the view that this increased metabolism is a result of stimulation by a toxic substance. Since a rise in blood urea is also found in such conditions as simply laparotomy (99), sterile abscess (23), pleuritis (23), pancreatitis (23), bilateral suprarenalectomy (82), and anaphylaxis (95), he is of the opinion that the reaction is rather non-specific in nature. It is a fact, also, that in the administration of fluid to an animal suffering from acute obstruction, the blood urea does not rise markedly. This, they have explained

on the basis of dilution of the toxin with a lessening of its effective activity.

Workers who explain the urea rise entirely on a basis of kidney impairment, Gamble and Ross (43), Brown, Eusterman, Hartman and Rountree (14), fail to report concomitant urinary findings and so their results cannot be considered too strongly.

Bacon, Anslow and Eppler (6) stated that in any condition causing dehydration there is a rise in blood urea. The dehydration, they explain, causes a loss of water from the protein molecule. This loss of water of hydration causes a disintegration of the protein molecule. The split products of the protein molecule are then metabolised, and as a result there is an immediate, and large rise in urea nitrogen. In the experimental proof of this theory, animals were dehydrated by the use of hypertonic salt solution. While this procedure produced dehydration, it also produced an increase in temperature, and so the experimental results lose their significance since there is no hyperpyrexia in intestinal obstruction. In fever, there is an abnormal disintegration of cellular protoplasm as can be demonstrated even microscopically.

It was the research work of MacCallum, Lintz, Vermilye, Leggett and Boas (64) which first demonstrated the changes which take place in the blood chlorides.

They found a lowering of the chlorides in intestinal obstruction.

Haden and Orr (50) found the diminution of the chlorides to be the first recognizable change which took place in the blood. It precedes the rise in urea. It is significant that the higher the obstruction, the more rapid is the loss of chlorides. The interrelationship of all the blood changes was shown when they found that by the subcutaneous administration of NaCl solution so as to maintain the blood chloride level constant the other blood changes did not occur. This was due almost entirely to the NaCl, they found. When isotonic glucose solutions, or sodium bicarbonate solutions were used to replace the fluid loss, the other blood changes quickly occurred, and death invariably followed. Also, administration of concentrated NaCl solution (10%) prevented toxemia, and the other blood changes.

By quantitative estimations of the chloride content of the urine and vomitus in experimental obstruction, Haden and Orr were unable to account for the chloride loss. Monkeys and rabbits - animals which do not vomit - were also observed to have a lowering of the blood chlorides. From this evidence, they evolved the theory of the specific detoxicating effect of the Cl^- ion. The hypothetical reaction they propounded was this:

$$\text{X (toxic)} + \text{NaCl} + \text{H}_2\text{CO}_3 \longrightarrow \text{NaHCO}_3 + \text{X (HCl)} \text{ (nontoxic)}$$

This nontoxic substance was supposed to be held in the

tissues and prevent the further action of the toxin in promoting protein destruction. There are several very strong arguments against this theory. In the first place, there is no other chloride salt which can be used to replace the NaCl. KCl, NH_4Cl , CaCl_2 , and MgCl_2 are all ineffective, and while this does not entirely break down the theory, it at least lessens its strength. Another fact, which they overlooked, and which is very important in explaining the obstructive effects in monkeys and rabbits, is the fact that all rabbits and monkeys which died from intestinal obstruction had greatly dilated stomachs. Fluid which is regurgitated into the stomach must be considered as being outside the body.

Sugito, (quoted by Cooper (24)), found that by dialyzing the toxic intestinal contents of an obstruction against isotonic NaCl he was able to destroy its toxicity.

Copher and Brooks, (25), and Gatch, Trusler and Ayers (44), weakened the theory of Haden and Orr when they reported that in cases of strangulation the administration of NaCl was ineffective.

Gatch, Trusler and Ayers (44) determined the chloride content of the urine, blood, vomitus, and tissues in cases of obstruction. The tissue chlorides were reduced in proportion to the blood chlorides. This cannot be

taken as positive evidence of the untenability of Haden and Orr's theory since there is no chemical method for fractionally determining the chemical combinations of the Cl^- ion in the tissues. According to their analyses, there was enough chloride loss in the urine and vomitus to account for all the chloride lost from the body. In animals which did not vomit, the chloride content of the fluid in the stomach and the urine accounted for all the blood chloride loss.

When the CO_2 combining power of the blood is disturbed, there is a disturbance of the efficiency of the acid-base regulating mechanism of the blood. Of all the buffers of the blood, the $\frac{\text{NaHCO}_3}{\text{H}_2\text{CO}_3}$ is the most readily affected.

Hastings, Murray and Murray (55) first demonstrated the increased CO_2 combining power of the blood in intestinal obstruction. Later Gamble and Ross (43) formulated their theory as to the ionic changes which occur in the blood with obstruction. They thought the failure of physiological processes and even the death following experimental obstruction of the pylorus could be explained on the basis of the continued reduction of the volume of body fluids. This loss was due to a demolition of their sustaining structure caused by a loss of Cl^- and more significantly of Na^+ in the vomited stomach secretions. The beneficial effect from introducing Na^+ and Cl^- into the body along with water is to

be regarded as being due simply to a repair of this structure permitting recovery of a normal volume of body water. The unique action of NaCl in affecting plasma repair can then be explained since it is the only salt which contains both the necessary ions Na^+ and Cl^- . The mechanism of the increased CO_2 combining power of the blood, then, is this. Following obstruction of the pylorus there occurs in the vomited stomach secretions a loss of Na^+ and Cl^- from the body. The sum of the concentrations of the acid radicals in the body fluids being determined, owing to the adjustability of HCO_3^- , by the fixed base concentration, a loss of Cl^- does not deplete the total ionic concentration whereas a loss of Na^+ does, and also removes an equivalence of HCO_3^- . Reduction of the ionic content of the body fluids by withdrawal of Na^+ is the significant factor in the rapid dehydration following pyloric obstruction. Dehydration cannot be repaired by the introduction of water alone (glucose solution), or of water and the chloride ion (NH_4Cl). The efficacy of NaCl solution in sustaining the usual volume of body fluids is due to replacement of the loss of Na^+ . The increase of HCO_3^- in the plasma, which tends to occur following pyloric obstruction, is an automatic consequence of the depletion of Cl^- . The degree of alkalosis is, however, usually greatly lessened by reduction of plasma base due to loss of Na^+ , by extension of the base equivalence of plasma protein caused both by an increase in concentration of protein and of

plasma alkalinity, and, possibly, by an increase in the concentration of organic acids. Following administration of NaCl solution, the surplus of Na^+ over Cl^- presenting for excretion owing to greater depletion of the latter is conveyed into the urine as bicarbonate.

Atchley and Benedict (3) in experimental intestinal obstruction were able to confirm the blood changes of previous investigators: (1) dehydration, (2) nitrogen retention, (3) loss of chloride, (4) increase in carbonate. They also found a decrease in serum base, and a relatively (to chloride) high loss of base in the vomitus. The increase in phosphate, sulfate, and protein anions easily accounts for the large increase in undetermined acid found by Gamble and Ross. It is, then, possible to say that the major factors governing the disappearance of serum chlorides in intestinal obstruction are: (1) loss of BCl and HCl in the vomitus (and urine), and (2) replacement of chloride by an increase in the protein, sulfate, and phosphate anions - due essentially to dehydration. It seems unlikely that there is place in such a mechanism for the neutralization by chloride of an hypothetical toxin as Haden and Orr suggest. In their experimental work, Atchley and Benedict found no consistent change in undetermined acids, and therefore no necessity for assuming changes in organic acid content. An analysis of the factors causing disappearance of the chlorides indicates the change is due to (1) loss of BCl and HCl by vomitus, and (2) replacement

of the base bound to chlorides by acids (phosphate and sulfate) retained because of renal insufficiency make it doubtful that this latter change is brought about by actual loss of chlorides from the body. It is tentatively suggested by Atchley and Benedict that the beneficial results of nonoperative treatment on the clinical course of intestinal obstruction are best followed by their effect on the dehydration of the blood as determined by serum protein per cent, hematocrit, and hemoglobin.

Alsina and Pyoan (2) found that varying the site of occlusion of the intestine caused different blood chemical changes to take place. High occlusion of the intestine in dogs was observed to cause an increase in the alkaline reserve, and a diminution of the chlorides. A low ileal occlusion also caused a fall of the chlorides, but no marked change of the alkaline reserve was noted. A temporary occlusion caused the same alterations, and recovery took place with the restoration of function. In strangulation of a high segment of intestine ten to twelve cm. long, the same changes were noted in the chloride content of the blood and the alkaline reserve. A strangulation of a low ileal segment was found to produce a hypochloremia, but no lowering of the alkaline reserve. These experiments again make evident that in strangulation of the intestine, toxemia is the predominant factor, and produces effects similar to, but

more rapidly than does a simple obstruction.

Dragstedt (32) has shown that a relationship exists between the blood chemical changes and the pressure within a closed loop. He found that if the distention in the closed segment be relieved by aspiration of the fluid, the toxemia is reduced, and there is a return of the blood chloride towards the normal. The intra-peritoneal injection into normal dogs of the fluid aspirated from a closed loop produces a toxemia with the results mentioned above - hypochloremia, rise in blood nonprotein nitrogen and urea.

Some further blood changes have been noted by Haden and Orr (51). With cardiac and pyloric obstruction, they noted a marked rise in the viscosity of whole blood, and some increase in plasma viscosity. Upper jejunal obstruction caused only slight changes. No blood concentration was noted, but the toxemia was found to parallel the increase in viscosity. No change was observed at any time in the surface tension of the blood. Blood sedimentation was found to be quicker than normal after cardiac, pyloric, and jejunal obstruction due evidently to a change in the physicochemical status of the blood.

From the time relationships, especially in strangulation, it is evident that the symptoms and death in intestinal obstruction are not due solely to blood chemical changes or to toxemia. It is a combination of the two

which produces the characteristic clinical picture.

2. Central Nervous System. No pathological changes have been observed in this system.

3. Heart and Lungs. These organs are normal.

4. Liver. Both Hartwell (54), and Whipple (84) have found that congestion and fatty degeneration are occasionally present.

5. Kidneys. Hartwell (54) has reported marked congestion in the kidney and the presence of granular coagula in the tubules. South and Hardt (81) found only cloudy swelling. On the other hand, Draper (36) and Whipple (84) have reported that the kidneys are normal.

6. Spleen. South and Hardt (81) found the spleen to be swollen in their cases of experimental intestinal obstruction.

TREATMENT.

A. Preventive.

Grover (48) has emphasized the use of a proper technique at the operating table in preventing intestinal obstruction. Excessively hot sponges may irritate both the bowel and the peritoneum so as to produce local reactions which will later cause obstructive symptoms. In a like manner, the exposure of the intestine to cold air should be reduced to a minimum, so as to reduce the chances of local reaction. Rough handling of the intestine or rubbing it with gauze may so traumatize it that postoperative adhesions will form readily, and possibly produce a stricture of the intestinal lumen.

B. Drugs and Surgery.

Carlson and Wangensteen (19) have shown that in intestinal obstruction the bowel distal to the point of obstruction is anatomically normal, while the part on the proximal side shows changes. Tracings of the movements of the distal loop closely resemble those of the normal. On the whole, they found greater activity in the distal than in the proximal loop. When using dogs as the experimental subjects, they found pituitrin caused either no response, or a depression of intestinal movements under the conditions of the experiment. The cause of the mode of action of pituitrin has been explained by Elmer, Ptaszek, and Scheps (40). In rabbits they found vasopressin augmented intestinal movements, and oxy-

tocin depressed them. Vasopressin was thought to be more active in stimulating the intestine than pituitrin owing to the presence in the latter of oxytocin. They also have reported several cases of paralytic ileus in which vasopressin was used with good effect.

Hartman (52) has advocated the use of choline in cases of paralytic ileus. He found that when it was injected intravenously peristalsis was promoted, although the action was very fleeting. Its use might be justified as an instigator, not a sustainer of intestinal movement in paralytic ileus.

The use of spinal anaesthesia with novocaine has been grossly abused in the treatment of intestinal obstruction. Markowitz and Campbell (66) produced paralytic ileus in dogs by the intraperitoneal injection of iodine or severe intra-abdominal trauma. In such cases where the intestinal obstruction was a result of a reflex paralysis of peristalsis, the spinal anaesthesia was of great help in curing the condition.

Following the work of Haden and Orr (50) the action of hypertonic NaCl solution in promoting intestinal peristalsis has been studied. Ross (76) found he got beneficial results in the experimental ileus of dogs by injecting 20 per cent NaCl solution intravenously.

Bouisset and Fabre (9) (10) in studying the physiological action of hypertonic NaCl solutions found

that the injection of such solutions at varying intervals after occlusion of the small intestine produced two distinct phases in the muscular tone of the intestinal wall. In the first phase, the wall is sluggish but ready to react violently under the influence of the excitant. The pressure within the intestine during this phase rose from 2.5 cm. Hg to 8 cm. Hg. In the second phase, atony is pronounced and the reactions are weak or negative. It requires five or six days of occlusion to show the complete change. From 10 to 20 cc. of 30 per cent NaCl solution were injected intravenously in those experiments in which there was an increased activity of the intestinal contractions.

Since the NaCl solution as used clinically has for its purpose the overcoming of dehydration as well as intestinal stimulation, it is best to use about a 2 or 3 per cent solution. This may be injected intravenously or subcutaneously. A total of 1 gm. of NaCl per kilo body weight may be given, although the administration should be discontinued when the blood chlorides return to normal, or there is evidence of "water-logging" of the tissues.

Gatch, Trusler and Ayres (45) have stressed the fact that ordinarily surgical intervention in intestinal obstruction is not an emergency procedure. The surgical treatment will depend on the type and duration of the causative factor. The operation should be delayed until

the patient's dehydration, hypochloremia and starvation are overcome by the use of intravenous glucose and intravenous NaCl solution. The general principles which must be borne in mind are these: (1) The toxemia is closely allied to surgical shock. So, the patient withstands a general anaesthetic very poorly. There is also little resistance to operative trauma. (2). The dehydration and hypochloremia must be combatted first. (3). Intestinal anastomosis is never to be undertaken in the presence of great distention of the bowel. The seminecrotic tissues cannot be sutured safely, and fresh surfaces are opened for the rapid absorption of toxic material. The paralytic ileus which remains prevents adequate evacuation of the intestine. As stated above, it is this paralysis of the intestinal musculature which causes the failure of an enterostomy.

In case a loop of intestine has become gangrenous, it should be excised and a "gunbarrel" enterostomy performed. When the patient has completely recovered from this operation, an intestinal anastomosis can be safely established.

The use of hot abdominal packs has been stressed by Orr. Whether the action they exert is direct or reflex is unknown, but he states that from experience he has learned they are useful in stimulating intestinal peristalsis, and maintaining the tonicity of the intestinal wall.

In postoperative cases, where there is a paralysis of both vagal and sympathetic systems, the aim of the treatment is to restore the power of contraction to the paralysed muscle. As with any other exhausted portion of the neuromuscular system, it is essential to provide rest for the prevention of the onset of the condition. The free use of morphine will do much to prevent the onset of such a condition. In the early stages it may be possible to bring about a cure by directly stimulating the muscle which is only slightly exhausted. Walton (93) advocates the administration of one drachm of 1:1000 suprarenal extract with one drachm of water every hour for six doses. In case the intestines, and not the stomach, are involved, eserine sulfate gr.1/200 may be administered hypodermically. The musculature cannot be rested while it is stretched, and, therefore, if dilatation is actually present the viscus must be emptied. Frequent gastric lavage is essential, or continuous gastric or duodenal drainage through a Levine tube may be employed. An enterostomy is not so satisfactory, for in these cases it is almost impossible to empty the whole length of the gut. If only one loop be affected, the gut should be drained from above. Any other form of operative treatment, such as lateral anastomosis, is almost certain to fail, since the wall of the gut is completely paralysed.

In cases with a relative paralysis of all vagal

impulses, Ritvo and Weiss (74) were able to demonstrate under the fluoroscope that eserine sulfate either hypodermically or by mouth gave almost immediate relaxation to the spasmodic area. This drug also increased peristalsis, and heightened the tonus, thus supporting the view that the changes are due to a relative weakness of the vagal impulses. If the spasm is confined to the pyloric sphincter, Deaver (29) advocates its free division. Gomain (47) has attempted cures by excision of the semi-lunar ganglion and the splanchnic nerves.

The group which is characterized by relative paralysis of the vagal impulses to the sphincters is best attacked surgically by forceful dilatation, and stretching of the sphincter muscle. The use of mercury bougies or hydrostatic dilators is good only in the very early stages of the condition. By the time the surgeon sees such a case, more drastic measures are usually necessary. Mikulicz (71) has devised an operation whereby the fingers may be introduced into the sphincter, and the stretching be accomplished in this manner. The dangers of such a procedure are self-evident - laceration and hemorrhage of the mucosa, postoperative fibrous stricture, or rupture of the gut wall with resultant sepsis.

If a pylorospasm is the result of extra-pyloric pathology, the correction of such pathology will almost invariably correct the reflex pylorospasm.

Until a few years ago, the treatment of Hirschsprung's disease was the surgical diversion of the intestinal contents from the affected portion of the gut. Ito and Soyesima (59) reported that colectomy gave the best operative results. Later surgeons - Bartle (8), Wade and Royle (92) - attempted to treat the condition by neurosurgical means. Lumbar ramisection has seemed to be very effective although the duration of the relief is still problematical.

The salient points in the treatment, then may be summarized as follows:

1. Prophylactic - by the use of proper surgical technique.
2. Gastric lavage - either continuous drainage, or lavage every six hours.
3. Morphine - to rest the intestine, and prevent it from tiring too soon.
4. Eserine sulfate and adrenalin - to maintain tonicity of intestinal musculature.
5. Spinal anaesthesia - to aid in production of a relative vagotonia.
6. Intravenous saline solution and intravenous glucose solution - to combat dehydration, hypochloremia, starvation, reduce toxemia, and increase elimination.
7. Enterostomy - for immediate drainage of a loop of intestine.
8. Intestinal resection - for elimination of a gangrenous section of gut.
9. Abdominal hotpacks - for stimulation of intestinal tone.

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